

In the Specification

Please substitute the following paragraph on page 9, beginning at line 18:

Figure 2 shows **FIGURES 2A-2I** show the result of Fluorescence-activated cell-sorter binding assays binding assay of control Ba/F3 cells and of T-cadherin expressing Ba/F3 cells, as explained in Example 4.2. FIGS. 2A to 2C correspond to FACS binding assay of control Ba/F3 cells, FIGS. 2D to 2I correspond to FACS binding assay of retroviral infected T-cadherin (pBI-GFP-Tcad) expressing Ba/F3 cells.

Please substitute the following paragraph on page 18, beginning at line 24 through page 19, line 10:

As used herein, the term “soluble form of T-cadherin” refers to a T-cadherin polypeptide that is not attached to the membrane. T-cadherin polypeptides that are not attached to the membrane can easily be generated by those of skill in the art by mutating the GPI-anchor site of a T-cadherin polypeptide. For example, the glycine at position 693 of SEQ ID NO: 1 may be changed to another amino acid. Alternatively, the soluble form may be a fragment of T-cadherin lacking the GPI-anchor site. Preferably, the soluble form of T-cadherin is selected from the group consisting of:

- a) a polypeptide consisting of amino acids 23 to 692 of SEQ ID NO: 1;
- ~~b) a polypeptide consisting of amino acids 23 to 692 of SEQ ID NO: 1;~~
- eb) a polypeptide consisting of a fragment of at least 50, 100, 150, 200, 250, 300, 350, 400, 450, 500, 550, 600 or 650 amino acids of (a) ~~or (b)~~;
- dc) a mutein of any of (a) to (eb), wherein the amino acid sequence has at least 80%, 90%, 95%, 96%, 97%, 98% or 99% identity to at least one of the sequences in (a) to (eb);
- ed) a mutein of any of (a) to (eb) which is encoded by a nucleic acid which hybridizes to the complement of a DNA sequence encoding any of (a) to (eb) under highly stringent conditions; and
- fe) a mutein of any of (a) to (eb) wherein any changes in the amino acid sequence are conservative amino acid substitutions of the amino acid sequences in (a) to (eb).

Please substitute the following paragraphs on page 36, beginning at line 19 through page 37, line 6:

The above cell lines were subsequently used for FACS binding assays, as shown on Figure 2. Panels 1 to 3 FIGS 2A-2C correspond to FACS binding assay of control Ba/F3 cells. Panels 4 to 9 FIGS 2D-2I correspond to FACS binding assay of retroviral infected T-cadherin (pBI-GFP-Tcad) expressing Ba/F3 cells. Binding was studied for:

- 0 nM (panels 1 and 4 FIG. 2A and FIG 2B), 6 nM (panels 2 and 5, 7-9 FIG. 2A, FIG 2E, FIG. 2G, and FIG. 2I), or 60 nM (panels 3 and 6 FIG. 2C and FIG. 2F) of 5'Flag-Acrp30 hexamer;
- 60 nM Acrp30 hexamer (panel 7 FIG. 2G);
- 10 mM EDTA (panel 8 FIG. 2H); and
- 10 µg/ml C1q (panel 9 FIG. 2I).

Control, uninfected Ba/F3 cells exhibited low binding to 6 nM (panel 2 FIG. 2B) or 60 nM (panel 3 FIG. 2C) 5'Flag-Acrp30 hexamer, while Ba/F3 cells expressing T-cadherin demonstrated increasing binding with 6 nM (panel 5 FIG. 2E, and panels 7-9 FIGS. 2G-2I) or 60 nM (panel 6 FIG. 2F) 5'Flag-Acrp30 hexamer (concentration expressed as trimer-equivalents). Background binding in the absence of ligand was low (panel 4 FIG. 2E). Including 60 nM of eukaryotic produced, untagged Acrp30 hexamer inhibited binding of 6 nM 5'FlagAcrp30, (panel 7 FIG. 2G), indicating specific binding between Acrp30 and T-cadherin. To examine the divalent cation requirements for binding, 10 mM EDTA were added to the binding reaction. This completely blocked binding (panel 8 FIG. 2H), indicating divalent cations are required for binding. C1q, a molecule that shares homology to Acrp30, did not affect binding at a 20-fold excess (by weight) when co-incubated with 6 nM 5'Flag-Acrp30 hexamer (panel 9 FIG. 2I), indicating that C1q likely does not bind to the same receptor as Acrp30.

Please replace original Figure 2 with the attached new Figure 2.